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Original Article

Intranasal corticosteroids for mild childhood obstructive sleep apnea – a randomized, placebo-controlled study

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ABSTRACT

Background: The use of non-surgical treatment for childhood obstructive sleep apnea (OSA) is gaining popularity, especially in children with mild disease.
Objective: To test the hypothesis that intranasal corticosteroids reduce disease severity in children with mild OSA.
Study design: A randomized, double-blinded, placebo-controlled trial of intranasal mometasone furoate (MF) versus placebo in children aged 6 to 18 years with mild OSA. The primary outcome was the change from baseline obstructive apnea hypopnea index (OAHI), as documented by overnight polysomnography, after four months of treatment.
Results: Sixty-two children were recruited but 12 dropped out. This left 24 and 26 children for final analysis in the MF and placebo group, respectively. The OAHI and oxygen desaturation index (ODI) improved significantly in the MF group only. The OAHI decreased from 2.7 ± 0.2 to 1.7 ± 0.3 in the MF group, but increased from 2.5 ± 0.2 to 2.9 ± 0.6 in the placebo group ($p = 0.039$). The mean changes in ODI in the MF group and placebo group were -0.6 ± 0.5 and $+0.7 \pm 0.4$, respectively ($p = 0.037$).
Conclusion: Four months of treatment with intranasal mometasone furoate effectively reduces the severity of mild OSA in children.

1. Introduction

Childhood obstructive sleep apnea (OSA) is characterized by prolonged partial and/or intermittent complete upper airway obstruction during sleep, which disrupts normal ventilation and sleep architecture [1]. The prevalence of childhood OSA in healthy children has been reported to be up to 5% [2,3]. If the condition is not treated, it can lead to a variety of important complications, namely: neurobehavioral deficits, systemic hypertension, and ventricular and endothelial dysfunction [4–6]. Neurocognitive deficits have also been reported, even in children with mild disease (obstructive apnea hypopnea index [OAHI] 1–5) [7].

Adenotonsillar hypertrophy is the most common cause of childhood OSA, and adenotonsillectomy remains the first-line treatment of choice; it can significantly improve the apnea hypopnea index [1,8]. A recent randomized trial showed that when compared with watchful waiting, adenotonsillectomy in school-age children with OSA resulted in greater reductions in symptoms and greater improvements in behavior, quality of life, and polysomnographic findings [9]. However, adenotonsillectomy may not be universally suitable for all children with OSA and relative contraindications include: very small tonsils and adenoid; bleeding disorders refractory to treatment; and submucus cleft palate or other medical conditions making patients medically unfit for surgery [1]. The procedure is not without risks, and complications such as hemorrhage and post-surgical respiratory compromise have been reported to occur in up to 28% of OSA patients [10,11]. The acceptance of surgery as treatment for sleep apnea in Hong Kong is generally low, and parents are always seeking non-surgical alternatives. Furthermore, there is no consensus on the cut-off of OSA severity for adenotonsillectomy, and it is usually reserved for children with moderate-to-severe disease, namely those with an OAHI >5/hr. At the time of writing, the management of children with the milder form of OSA has not been unified; however, the use of non-surgical treatment options is gaining popularity.

Nasal and oropharyngeal inflammation is present in children with OSA and may play an important role in the pathogenesis of

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breathing disturbances during sleep [12]. Local and systemic inflammatory markers and pro-inflammatory cytokines are increased in children with OSA and promote lymphoid tissue proliferation [13]. Therefore, anti-inflammatory agents, especially topical nasal spray corticosteroids, are suggested to have a potential role in reversing adenotonsillar enlargement [14–16]. Furthermore, these medications have proven effectiveness against allergic rhinitis, which is a common co-morbidity in children with OSA [2]. A recent Cochrane review suggested the necessity for further randomized, controlled trials in order to evaluate the use of topical nasal corticosteroids in children with OSA [3].

The present study was a randomized, double-blinded, placebo-controlled trial to test the hypothesis that topical intranasal corticosteroids reduce disease severity in children with mild OSA. The primary endpoint of interest was the change from baseline OAHI, as documented by overnight polysomnography (PSG), after four months of treatment. The secondary endpoints included the change from baseline in: (1) tonsil and adenoid size; and (2) nasal symptoms. It was hypothesized that treatment with intranasal corticosteroids would lead to a significant reduction in OAHI.

2. Methods

2.1. Subjects and study design

Children aged 6–18 years, who attended the sleep disorder clinic and reported to have symptoms of sleep disordered breathing (SDB), were recruited from May 2006 to March 2008 for overnight PSG. Suitable subjects were invited to participate in the study if they had habitual snoring (≥3 nights per week) and their PSG revealed mild OSA (OAHI of ≥1–5). Exclusion criteria were the presence of any of the following: genetic syndromes, congenital or acquired neurological diseases, neuromuscular diseases, craniofacial abnormality, previous upper airway surgery, known fixed nasal obstruction such as previous nasal fracture or deviated nasal septum.

Restricted block randomization was implemented. The children were allocated to receive mometasone furoate (MF) or placebo, using double-blinded randomization with a sealed opaque envelope method. The allocation was based on a computer-generated randomization list with varying block sizes. The procedures of randomization and allocation were performed by one of the investigators (AM Li), who was not involved in the data collection. Corticosteroids (MF) and placebo were provided in numbered identical containers. The appearances of the active drug and placebo were indistinguishable. Each corticosteroid spray delivered 50 mcg of active drug. The study drug was given as two sprays per nostril in the evening for the subsequent four months. The dose was chosen as it has been shown to be safe and effective for children with allergic rhinitis [17]. The participants were not allowed other medications that could influence nasal inflammation or patency, such as leukotriene receptor antagonist, antihistamines, and decongestants. Before the start of treatment, each child had completed a symptom questionnaire and had undergone an upper airway evaluation by an otorhinolaryngologist. All participants underwent PSG, upper airway examination, and questionnaire assessment again at the end of a 4-month treatment period. Each participant was also given a diary to complete on a daily basis in order to ensure therapy compliance and to document any side effects from the medication.

This study was approved by the Joint Chinese University of Hong Kong – New Territories East Cluster Clinical Research Ethics Committee and Centre for Clinical Trials, The Chinese University of Hong Kong (reference number: CUHK_CCT00119). Informed consent and assent were obtained from the legal caregiver of each child and the child, respectively.

2.2. Overnight polysomnography evaluation

The overnight PSG was performed in a dedicated sleep laboratory with Siesta ProFusion II PSG machine (Compumedics, Australia) [18]. The following parameters were recorded: electroencephalogram (EEG) from four channels (C3/A2, C4/A1, O1/A2, O2/A1), bilateral electro-oculogram, electromyogram (EMG) of mentalis activity, and bilateral anterior tibialis. Piezo respiratory effort belts measured respiratory movements of the rib cage and abdomen. Electrocardiogram and heart rate were continuously recorded from two anterior chest leads. Arterial oxyhemoglobin saturation (SaO2) was measured by an oximeter (Ohmeda Biox 3900 Pulse Oximeter; Ohmeda, Louisville, CO) with a finger probe. Respiratory airflow pressure signal was measured via a nasal catheter placed at the anterior nostril nares and connected to a pressure transducer. A snoring microphone placed near the throat measured snoring. Body position was monitored via a body position sensor.

OSA was defined as absence of airflow with persistent respiratory effort lasting at least two baseline breaths, irrespective of SaO_2 changes. Mixed apnea was defined as the absence of airflow for a duration of at least two breath cycles without inspiratory effort in the initial portion of the event, followed by resumption of inspiratory effort in the second portion of the event. Obstructive hypopnea was defined as a reduction of 50% or more in the amplitude of the airflow signal with persistent respiratory effort. It was only quantified if it was at least two baseline breaths and was associated with oxygen desaturation of at least 3% and/or arousals. OAHI was defined as the total number of obstructive apneas, mixed apneas and obstructive hypopneas per hour of total sleep time. Oxygen desaturation index (ODI) was defined as the total number of dips in arterial oxygen saturation at least 3% per hour of sleep. Arousal was defined as an abrupt shift in EEG frequency during sleep, which may include theta, alpha and/or frequencies greater than 16 Hz, but not spindles, with 3–15 s in duration. During REM sleep, arousals were scored only when accompanied by concurrent increases in submental EMG amplitude. Arousal index (ArI) was defined as the total number of arousal per hour of total sleep time.

The PSG data were scored and interpreted by an experienced sleep technologist who was blinded to the subjects' group allocation.

2.3. Upper airway examination

The size of tonsils and adenoid was examined endoscopically by means of a flexible fiberscope (Olympus 3 mm; Olympus, Japan). An otorhinolaryngologist, who was blinded to the group allocation and PSG result of the participants, performed the examination. Tonsil and adenoid size were reported as percentages of the oropharyngeal and nasopharyngeal airway and graded according to the following system (Table 1) [19].

2.4. Nasal symptom score and frequency of snoring

Nasal symptoms during the past month were assessed before and after the intervention with a visual analog scale ranging from 0 to 10 for each of the following four components: nasal blockage, nasal discharge, nasal pruritus, and eye pruritus. A composite score was

Table 1
Grading of adenoid and tonsil size.

Grade	Percentage of airway occupied by soft tissue
1	0–25%
2	26–50%
3	51–75%
4	76–100%

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Table 2
Subject characteristics before and after intervention.

	Intranasal mometasone furoate group ($n = 24$)			Placebo group ($n = 26$)			p_1	p_2
	Pre	Post	p^*	Pre	Post	p^*		
Age, yr	10.7 ± 0.6	11.1 ± 0.6	<0.0001	11.5 ± 0.5	11.9 ± 0.5	<0.0001	0.30	0.20
Gender, female/male	9/15			7/19			0.42	
Height, cm	142 ± 3	144 ± 3	<0.0001	147 ± 3	149 ± 3	<0.0001	0.29	0.58
Weight, kg	43 ± 3	44 ± 3	<0.0001	50 ± 3	53 ± 3	0.011	0.09	0.41
Body mass index, kg m^{-2}	20.7 ± 0.9	20.8 ± 1.0	0.39	23.0 ± 1.1	23.4 ± 1.0	0.29	0.11	0.57
Body mass index *z* score	1.09 ± 0.22	1.03 ± 0.23	0.21	1.27 ± 0.26	1.37 ± 0.23	0.44	0.59	0.25
Allergic rhinitis, %	79.2			80.8			0.89	
ODI, hr^{-1}	1.7 ± 0.3	1.1 ± 0.2	0.049	1.8 ± 0.3	2.5 ± 0.6	0.19	0.84	0.037
SpO_2 nadir, %	90 ± 0.6	91 ± 0.6	0.48	89 ± 0.5	90 ± 0.7	0.025	0.025	0.23
OAHI, hr^{-1}	2.7 ± 0.2	1.7 ± 0.3	0.013	2.5 ± 0.2	2.9 ± 0.6	0.45	0.43	0.039
Arousal index, hr^{-1}	10.2 ± 1.0	13.1 ± 2.3	0.17	8.9 ± 1.4	10.6 ± 1.1	0.22	0.48	0.60
REM sleep, %	20.7 ± 0.9	21.5 ± 1.1	0.53	21.7 ± 0.9	21.5 ± 0.8	0.83	0.41	0.52
Slow wave sleep, %	31.6 ± 1.4	32.1 ± 1.9	0.72	32.4 ± 1.9	31.5 ± 2.1	0.58	0.75	0.52
Nasal blockade (0–10)	3.4 ± 0.5	2.8 ± 0.5	0.21	5.0 ± 0.5	4.5 ± 0.4	0.28	0.033	0.87
Nasal discharge (0–10)	3.0 ± 0.6	3.3 ± 0.6	0.62	3.6 ± 0.6	3.5 ± 0.6	0.84	0.4	0.64
Nasal Pruritus (0–10)	3.3 ± 0.7	2.5 ± 0.6	0.28	2.8 ± 0.6	3.0 ± 0.6	0.84	0.88	0.37
Eye Pruritus (0–10)	2.9 ± 0.7	1.9 ± 0.5	0.18	3.3 ± 0.7	2.9 ± 0.5	0.53	0.85	0.61
Composite (0–40)	12.6 ± 1.7	10.4 ± 1.5	0.19	14.8 ± 1.6	13.8 ± 1.5	0.53	0.34	0.61
Habitual snoring, %	75	54.5	0.031	50	50	1.00	0.07	

Abbreviations: p^*, obtained from paired *t*-test or McNemar test for within-group differences. p_1, obtained from independent *t*-test or Chi-squared test for baseline comparisons. p_2, obtained from 2-way ANOVA for comparison between intranasal mometasone furoate group and placebo group. OAHI, obstructive apnea hypopnea index; ODI, oxygen desaturation index; REM, rapid eye movement; SpO_2, oxygen saturation.

calculated by summation of the scores obtained from the four symptom components. Frequency of snoring in the past month was assessed based on parental reporting.

2.5. Sample size calculation

Local pilot data on the effect of intranasal corticosteroids in children with mild OSA were not available; therefore, sample size calculation was based on an assumption of an effect size of 0.8 (Cohen's D). Twenty-five subjects in each arm would be required to detect such difference with 80% power at a type 1 error rate of 5%. With an estimation of 20% dropout rate, this study aimed to recruit 31 subjects in each arm.

2.6. Outcome variables and statistical analysis

The primary outcome variable was change in OAHI. Secondary outcome variables were changes in tonsil and adenoid size, and nasal symptoms. Results were presented as mean values (±standard error of mean), unless otherwise specified. Baseline comparisons were made by independent *t*-tests and Chi-squared tests for continuous and categorical data, respectively. Continuous data were subjected to statistical analyses using paired *t*-tests and 2-way analysis of variance procedures for repeated measures to test for within-group and between-group differences, respectively. Chi-squared test was used to test for between-group differences in proportions. McNemar and marginal homogeneity tests were used to test for within-group differences in dichotomous and categorical data, respectively. A 2-tailed $p < 0.05$ was considered to be statistically significant. All the analyses were performed using the statistical software packages SPSS (version 13.0 for Windows; SPSS Inc., Chicago, Illinois, USA).

3. Results

A total of 162 children were screened, of whom 62 were recruited and randomized to receive MF ($n = 31$) or placebo ($n = 31$). Of these, 12 children dropped out, resulting in 24 and 26 subjects in the MF group and placebo group, respectively, for final analysis (Fig. 1). The final study group had a mean (SD) age of 11.1 (2.8) and a mean (SD) OAHI of 2.6 (1.0).

A total of 10 adverse events were reported, including: nasal bleeding ($n = 6$, three in the MF group and three in the control group); nasal discomfort ($n = 2$, one in the MF group and one in the control group); throat discomfort ($n = 1$, in the MF group); vomiting ($n = 1$, in the MF group); and diarrhoea ($n = 1$, in the placebo group). A total of five subjects withdrew from the study because of these adverse events (Fig. 1). The baseline characteristics of the two study arms were not significantly different except for a slightly lower SaO2 nadir (89 ± 0.5 in the placebo group versus 90 ± 0.6 in the MF group, $p = 0.025$) and a higher nasal blockade score (5.0 ± 0.5 in the placebo group versus 3.4 ± 0.5 in the MF group, $p = 0.033$) in the placebo group (Table 2). Compliance rates were comparable between the two groups (90 ± 11.4% in MF group and 88.4 ± 16.1% in the placebo group, $p = 0.72$).

The OAHI and ODI improved significantly in the MF group only. The mean changes in OAHI in the MF group and the placebo group were –1.0 ± 0.5 and +0.4 ± 0.4, respectively ($p = 0.039$). The mean changes in ODI in the MF group and placebo group were –0.6 ± 0.5 and +0.7 ± 0.4, respectively ($p = 0.037$). The proportion of children having habitual snoring also reduced from 75% to 54.5% ($p = 0.031$) in the MF group but not in the placebo group. No significant between-group differences could be found in body size, daytime nasal symptoms, and sleep architecture (Table 2). The size of tonsils and adenoids were not significantly different between groups (Table 3). Treatment response was similar in subjects with

Table 3
Upper airway measurements before and after intervention.

	Intranasal mometasone furoate group ($n = 24$)			Placebo group ($n = 26$)			p_1
	Pre	Post	p^*	Pre	Post	p^*	
Tonsil size, %							
Grade 1	45.8	41.7	0.71	53.8	53.8	0.21	0.05
Grade 2	20.8	29.2		30.8	34.6		
Grade 3	25.0	25.0		0.0	7.7		
Grade 4	8.3	4.2		15.4	3.8		
Adenoid size, %							
Grade 1	54.2	56.5	0.32	46.2	57.7	0.13	0.77
Grade 2	25.0	26.1		30.8	23.1		
Grade 3	8.3	4.3		15.4	15.4		
Grade 4	12.5	13.0		7.7	3.8		

Abbreviations: p^*, obtained from marginal homogeneity test for within-group differences. p_1, obtained from Chi-squared test between the baseline characteristics of intranasal mometasone furoate group and placebo group.

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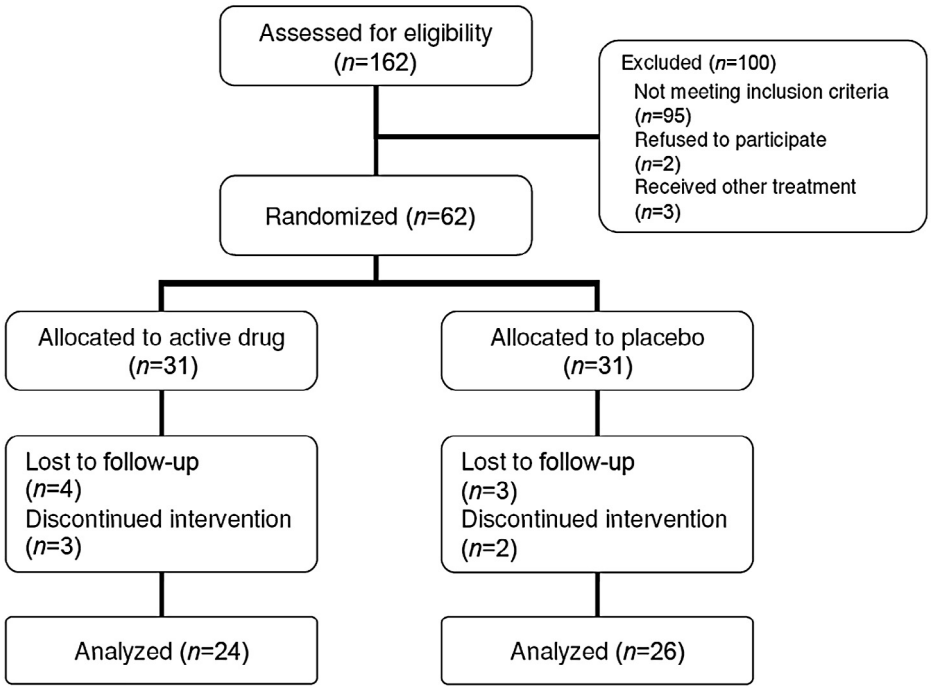

Fig. 1. Flow diagram of the subject recruitment and randomization.

smaller adenoid (grade I) and larger adenoid (grade II or above) at baseline (Table 4). Baseline adenoid size did not appear to predict treatment response. The majority of the subjects in both MF and placebo groups had allergic rhinitis (79.2% and 80.8%, respectively). In subgroup analysis, significant differences in change in OAHI between MF and placebo group were only demonstrated in the subgroup with allergic rhinitis, but not in those without allergic rhinitis (Tables 5 and 6).

4. Discussion

The present study demonstrated the beneficial effects of topical intranasal corticosteroids in mild childhood OSA. Both OAHI and ODI, together with a proportion of subjects with habitual snoring, significantly reduced after four months of treatment with intranasal mometasone furoate.

There is still much controversy in deciding when and how to treat childhood OSA. The lack of a standardized diagnostic definition, as well as the undetermined correlations between long-term clinical outcomes and different severities of the disease, are the major reasons behind the controversy. In children, an OAHI ≥1 has been shown to be statistically abnormal and this cut-off has been widely adopted in both clinical and research aspects; however, its clinical significance is still unclear [20]. In a previous study on the natural history of mild OSA, a modest improvement in OAHI over the course of two years was demonstrated, which suggested that watchful waiting may be an acceptable management option in some children with mild OSA.

Table 4
Changes in obstructive apnea hypopnea index (OAHI) in subgroup with different baseline adenoid size.

Adenoid grade I								
	Intranasal mometasone furoate group (n = 13)			Placebo group (n = 12)			p_1	p_2
	Pre	Post	p^*	Pre	Post	p^*		
OAHI, hr^{-1}	2.8 ± 0.3	2.0 ± 0.8	0.10	2.9 ± 0.3	3.5 ± 0.7	0.45	0.96	0.16
Adenoid grade II or above								
	Intranasal mometasone furoate group (n = 11)			Placebo group (n = 14)			p_1	p_2
	Pre	Post	p^*	Pre	Post	p^*		
OAHI, hr^{-1}	2.6 ± 0.3	1.4 ± 0.6	0.07	2.0 ± 0.3	2.2 ± 0.7	0.80	0.15	0.17

Abbreviations: p^*, obtained from paired t-test or McNemar test for within-group differences. p_1, obtained from independent t-test or Chi-squared test for baseline comparisons. p_2, obtained from 2-way ANOVA for comparison between the changes in obstructive apnea hypopnea index in intranasal mometasone furoate group and placebo group.

Table 5
Changes in obstructive apnea hypopnea index (OAHI) in subgroup with allergic rhinitis.

	Intranasal mometasone furoate group (n = 19)			Placebo group (n = 21)			p_1	p_2
	Pre	Post	p^*	Pre	Post	p^*		
OAHI, hr^{-1}	2.9 ± 0.2	1.9 ± 0.6	0.07	2.4 ± 0.2	3.1 ± 0.6	0.18	0.15	0.043

Abbreviations: p^*, obtained from paired t-test or McNemar test for within-group differences. p_1, obtained from independent t-test or Chi-squared test for baseline comparisons. p_2, obtained from 2-way ANOVA for comparison between the changes in obstructive apnea hypopnea index in intranasal mometasone furoate group and placebo group.

Table 6
Changes in obstructive apnea hypopnea index (OAHI) in subgroup without allergic rhinitis.

	Intranasal mometasone furoate group (n = 5)			Placebo group (n = 5)			p_1	p_2
	Pre	Post	p^*	Pre	Post	p^*		
OAHI, hr^{-1}	2.1 ± 0.5	1.1 ± 1.1	0.36	2.8 ± 0.5	2.0 ± 1.1	0.46	0.25	0.72

Abbreviations: p^*, obtained from paired t-test or McNemar test for within-group differences. p_1, obtained from independent t-test or Chi-squared test for baseline comparisons. p_2, obtained from 2-way ANOVA for comparison between the changes in obstructive apnea hypopnea index in intranasal mometasone furoate group and placebo group.

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However, 29% of children worsened over time, with younger age, male gender, and large tonsils being the risk factors for progression [21]. This raises the issue that although adenotonsillectomy is the first-line treatment, it may not be universally indicated and beneficial for all children with OSA. While there is growing evidence showing that even mild disease could lead to neurobehavioral and cardiovascular complications, whether early treatment could provide benefits and the most suitable treatment option remain important unresolved issues.

The use of intranasal corticosteroid therapy in childhood OSA is not new. Brouillette et al. conducted a triple-blinded, randomized, controlled trial in 25 children with a varying severity of OSA. They documented a significant reduction of OAHI in the group treated with 6-weeks of intranasal fluticasone [14]. The study also showed significant improvements in the frequencies of hemoglobin desaturation and respiratory movement/arousals, but no significant differences were demonstrated in adenotonsillar size or symptom score. Alexopoulos et al. published an open-labeled study with 27 subjects using four weeks of intranasal budesonide [15]. This study showed a reduction in mean OAHI and oxygen desaturation. There was also an improvement in the symptom score, and the effect could be maintained for nine months after completion of treatment. In a double-blinded, randomized, crossover controlled trial, Kheirandish-Gozal and Gozal reported that the use of intranasal budesonide for six weeks could significantly improve polysomnographic measures, magnitude of respiratory disturbance as well as adenoidal size. This study also demonstrated that the beneficial effect could be sustained for eight weeks after discontinuation of treatment [16]. However, the imbalance of the OAHI between the study groups at baseline, and possible carry-over effect, limited the validity of the results [3]. All the above studies consistently demonstrated the effectiveness of intranasal corticosteroid therapy in alleviating the severity of respiratory disturbances during sleep in children with OSA. However, they were limited by some intrinsic factors such as open-labeled design, short treatment duration and inclusion of subjects with varying OSA severity. A recent Cochrane review called for further randomized, controlled trials to evaluate the use of topical nasal corticosteroids in children with OSA [3].

The beneficial effect of intranasal corticosteroids is generally attributed to the reduction of inspiratory upper airway resistance at the nasal and adenotonsillar levels, although the exact mechanism has yet to be established. A study demonstrated that after 24 weeks of treatment with topical nasal beclomethasone, there was an 82% reduction in nasal obstruction symptom score and a 29% reduction in adenoid/choana ratio [22]. Topical corticosteroid therapy may provide benefits by exerting its anti-inflammatory effect on enlarged lymphoid tissue [3]. This hypothesis is supported by the findings of nasal and oropharyngeal mucosal inflammation and high abundance of glucocorticoid receptor in lymphadenoid tissues in the upper airway of children with OSA [23,24]. A significant reduction has been reported of a proinflammatory cytokine interleukin-6 in adenoid tissue in children with OSA treated with intranasal corticosteroids [25]. Leukotriene modifiers have also been considered as a therapeutic intervention in children with OSA. Their efficacy in reducing the severity of OSA and size of adenoid tissues was demonstrated by a recent randomized, controlled trial using 12 weeks of daily oral montelukast, which futher supports the role of anti-inflammatory agents as a non-surgical alternative for children with OSA [26].

The present study could not demonstrate significant changes in the size of tonsils and adenoids or symptom score. One of the possible explanations may relate to the subjective and crude grading method of adenotonsillar size used in the study. The scale might not be able to detect small but significant changes in the size of tonsils and adenoids adequately. Moreover, as subject recruitment and evaluation were carried out at different time points of the year, changes in adenotonsillar size, and severity of OSA might also be affected by natural seasonal variation due to varying prevalence of allergic triggers and respiratory viral infections throughout the year [27].

It is important to address several limitations of the present study. First, the study did not have a maintenance period and, therefore, could not provide data on the duration of treatment effect after cessation of medication. Moreover, clinical outcomes such as the effects on cardiovascular and neurocognitive function were not evaluated. This study found that intranasal corticosteroids appeared to be more effective in reducing OAHI in the subgroup with allergic rhinitis. However, whether the treatment effect depends on the presence of allergic rhinitis is still uncertain, as the subgroup analysis was underpowered. Allergic rhinitis is a prevalent condition: in the cohort in the present study, around 80% of children had allergic rhinitis. Poorly controlled allergic rhinitis could result in nasal obstruction that may lead to increased nasal airflow resistance and contribute to the development of sleep-disordered breathing [28–30]. Although the results did not show significant improvement in nasal symptoms with the use of intranasal corticosteroid therapy, the improved OAHI observed in children with allergic rhinitis after intranasal steroids suggests that alleviation of nasal symptoms and obstruction might be important while managing children with OSA and allergic rhinitis. Further studies are needed to investigate whether allergic rhinitis is the factor determining the effectiveness of intranasal corticosteroids in mild childhood OSA. The present findings show that OAHI, though improved, was not normalized, despite four months of treatment with intranasal corticosteroids. While there is growing evidence demonstrating long-term cardiovascular and neurobehavioral complications, even with mild OSA, the residual disease in the present cohort of children who received topical nasal steroid therapy warrants the necessity of further investigations to assess the long-term clinical outcomes and effectiveness of this form of OSA treatment.

5. Conclusion

The present study identified the effectiveness of four months of treatment with intranasal mometasone furoate in reducing the severity of mild OSA in children. These findings justify the use of topical steroids as a therapeutic option in children with mild OSA. Further studies with longer follow-up periods and evaluation of cardiovascular and neurocognitive complications are necessary to further delineate the effectiveness of intranasal corticosteroid therapy.

Conflict of interest

The study medication, MF, and placebo were supplied by Schering-Plough. The study was designed independently. The execution of the study protocol, data collection, analysis and interpretation, manuscript preparation, and decision to submit the paper for publication were conducted solely by the investigators. Dr Wing has received honorarium by serving as a part-time paid consultant for Renascence Therapeutics.

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The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2014.10.015.

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